Transcript Details

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Understanding Accelerated Biological Aging in Pediatric MS

ReachMD Announcer:
You're listening to *NeuroFrontiers* on ReachMD. On this episode, Dr. Jennifer Yang will discuss her research on biological aging in pediatric onset multiple sclerosis, or MS, which she presented at the 2026 ACTRIMS Forum. Dr. Yang is an Assistant Professor of Neurosciences at the UC San Diego School of Medicine and the Division of Pediatric Neurology at Rady Children's Hospital. Let's hear from her now.

Dr. Yang:
Our study is actually the first to examine the association of biological aging markers with clinical outcomes in MS. There's evidence that biological age may be a more precise way of measuring the effects of aging. In MS, we know that both chronological age and biological age are associated with disease expression as well as severity.

Two really well-recognized biological markers of the aging process are the shortening of telomeres and epigenetic age. In adult MS, we observe both deleterious associations of chronological age and these biological aging markers with disease severity. In addition to normal aging that may happen to all of us, people living with MS appear to also have accelerated biological aging.

In adults, this could be also confounded by the side effects of disability itself from the disease and other comorbid illnesses that they may be living with.

So, in order to avoid these confounders, our team, through the US network of pediatric MS centers, previously looked at these aging markers—the telomere length and epigenetic age in kids with MS. We know kids should not express evidence of normal aging or aging-related illnesses because they're pediatric patients. Unfortunately, in our previous publications, we've shown that kids living with MS also have accelerated aging. So, there's evidence that accelerated aging is most likely related to MS for pediatric patients.

In this study, while it's not proven, we think that the mechanism may be due to the burden of inflammation as well as the oxidative stress and cellular turnover that happen due to MS. And we know that all of this happens at an even higher level in children with MS because they have three to five times as many relapses as adults.

We know from adult MS studies that biological aging markers such as telomere length are strongly associated with clinical disability outcomes. In our current study in pediatric patients, we did not see the same association. There could be a couple of different explanations for this. One of them is the additional protective factors that youth have against neural dysfunction. In addition, while kids have greater biological age than birthdate-age-similar controls, as we have demonstrated in our previous studies, they still have not reached the levels of biological age seen in adults with MS just because they are younger.

We also know that our traditional outcome measures are still fairly crude and require substantial neuronal injury to measure clinically. It's possible that our kids with advanced biological ages do have more injury than others; we're just not seeing it beneath the surface using our current tools. However, this may catch up with them through mid-adulthood when more normal aging has also accumulated.

So, as expected in our study, we observed low levels of disability in pediatric patients using traditional disability measurements such as the EDSS. While pediatric MS patients demonstrate early accumulated aging damage, the lack of any association between aging markers and disability and relapse suggests that children who do not express progressive forms of disease also do not demonstrate associations of biological age with severe outcomes.

However, the consequences of accumulated aging damage may be delayed. We know that while children take longer to develop secondary progressive disease compared to adults, they achieve higher disability milestones at earlier chronological ages. One

explanation is potentially the delayed consequences of accelerated biological aging due to MS that start in childhood but catch up to them in early adulthood. So, overall, we think this presents unique opportunities for future research looking at the role of biological aging and MS across the lifespan.

ReachMD Announcer:
That was Dr. Jennifer Yang discussing biological aging in pediatric onset multiple sclerosis. To access this and other episodes in our series, visit *NeuroFrontiers* on ReachMD.com, where you can Be Part of the Knowledge. Thanks for listening!